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Renal and hepatic dysfunction parameters correlate positively with gender among patients with recurrent malaria cases in Birnin Kebbi, Northwest Nigeria

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Abstract

Background: Simultaneous increase in transaminases and bilirubin is an indicator of hepatic dysfunction in malaria. Malaria-induced hyperbilirubinemia has been associated with acute kidney injury and pathogenesis of cerebral malaria which are significantly associated with mortality in malaria infection. This retrospective study was designed to assess the lipid profile, and hematological, renal and hepatic function data of malaria patients in Sir Yahaya Memorial hospital Birnin Kebbi from 2016 to 2020 who are 18 years and above.

Methods: The data of all patients between 2016 and 2020 who are 18 years and above were collected. Complete data of 370 subjects who met the inclusion criteria which consist of 250 malaria subjects and 120 control subjects were analyzed.

Results: The results showed that females constitute 65.2% of malaria patients with complete records while the remaining 34.8% were males. Age distribution of the patients showed that the infection was more prevalent among 26–45 years and least among 65 years and above. Anemia and thrombocytopenia were prevalent among the female malaria patients compared to the male patients. Liver and kidney function parameters analyzed correlate positively with the gender. The infected male showed higher dysfunction in liver parameters while infected female patients showed significant dysfunction in kidney function parameters and lipid profile.

Conclusions: In conclusion, to prevent the potential widespread of acute renal and hepatic failure with the attendant morbidity and mortality among malaria patients, it is recommended that liver and kidney function tests be mandated for patients with recurring malaria and those with a history of treatment failure in the endemic area to ensure early diagnosis of malarial induced kidney and liver injury among malaria patients.

Highlights

- Liver and kidney dysfunction correlates positively with the gender.
- Infected male showed higher dysfunction in liver while female showed significant dysfunction in kidney

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- Anaemia and thrombocytopenia were prevalent among the female malaria patients compare to the male
- There may be potential widespread of acute renal and hepatic if liver and kidney function tests are not mandated for patients with recurring malaria

Keywords: Acute kidney injury, Anemia, Hepatic injury, Recurrent malaria, Thrombocytopenia

Background

Malaria is an infectious disease of poverty of public health concern in subtropical and tropical regions [1]. Around 241 million cases of malaria with 627,000 deaths were reported in 2020 compared to 227 million cases in 2019. This represents an increase of around 14 million with an increase of 69,000 deaths above what was reported in 2019 [2]. About 90% of all the reported cases of malaria and deaths occur in Africa making Africa the epicenter of this disease [1, 3, 4]. It is of importance to note that one quarter of the malaria cases in Africa with the attendant mortality and morbidity occur in Nigeria [5]. If malaria infection is not treated on time, malaria parasites might obstruct blood flow to vital organs [6], invade and destroy vital organs [3], and induce biochemical and metabolic changes that may be detrimental to the host [7].

Malaria-induced hepatic injury is known to be a major feature of malaria and a major cause of morbidity and mortality in malaria patients [8–10]. Simultaneous increase in transaminases and bilirubin is considered as an indicator of liver dysfunction in malaria. Malaria-induced hyperbilirubinemia is associated with acute kidney injury and pathogenesis of acute renal failure among malaria patients [11–15]. This suggests that hepatic dysfunction is closely related to the development of acute kidney injury. In addition to this, malaria-induced hyperbilirubinemia has been implicated in the pathogenesis of cerebral malaria which is significantly associated with mortality in malaria infection, making it a serious concern in malaria patients [12, 16–18]. Malaria-induced hepatic damage impairs lipid homeostasis leading to alterations in host lipid and lipoprotein profiles which associate with the risk of cardiovascular disease in malaria patients [7, 19, 20]. This retrospective study of the clinical data of malaria patients with recurrent malaria cases attending Sir Yahaya Memorial Hospital Birnin Kebbi between 2016 and 2020 was conducted to determine the effect of malaria on the lipid profile, hematological, and renal and hepatic function parameters of the patients in order to prevent the occurrence of malaria-mediated liver and kidney injury in malaria patients.

Methods

Study area, study population, data collection, and preparation

The study was carried out in Birnin Kebbi Metropolis (12° 27′ 57.8808″ N and 4° 11′ 58.2864″ E.), Kebbi State in the Northern part of Nigeria. The data of all patients attending Sir Yahaya Memorial Hospital Birnin Kebbi, Kebbi State, between 2016 and 2020 who are 18 years and above were collected. The data of 370 subjects who met the inclusion criteria with complete lipid profile, renal, and hepatic data consisting of 250 malaria subjects and 120 control subjects were used for this study. Data were obtained directly from the medical record unit and the patient's laboratory registration logbook using prepared data extraction sheet with the help of laboratory personnel. The data obtained include reporting date, age, sex, and laboratory results. Personal information of the patients such as name and address was not collected.

Exclusion criteria

Patients with pre-existing hepatic diseases, renal diseases, hypertension, human immunodeficiency virus, diabetes mellitus, acquired immune deficiency, viral hepatitis, drug-induced increase in liver and kidney biomarkers, and any condition that can affect the lipid profile, and all biomarkers being studied were not included in the study.

Ethical approval

Ethical approval was obtained from the hospital management before the commencement of data collection. The risk of loss of confidentiality was eliminated by the removal of personal identifiers and the use of identification numbers. All ethical procedures involving the use of human subjects under clinical settings were duly followed. The retrospective data was collected after ethical approval (SYMHBK/SUB/17/VOL.III/196) was obtained.

Statistical analysis

All statistical analyses were performed using GraphPad prism version 6 for Windows (GraphPad Software: San Diego, California, USA). The results were expressed as mean ± standard deviation. The differences in parameters across the groups were investigated using a one-way analysis of variance (ANOVA) followed by Tukey's

Table 1 Gender profile of patients with recurrent malaria cases in sir Yahaya memorial hospital Birnin Kebbi from 2016 to 2020

	Gender	Sample number	Percentage (%)
Control	Male	53	44.17 %
	Female	67	53.83 %
Infected	Male	87	34.80%
	Female	163	65.20%

Table 2 Age profile of patients with recurrent malaria cases in Sir Yahaya Memorial Hospital Birnin Kebbi from 2016 to 2020

Age	Frequency	Percentage
18–25	43	17.2
26–45	101	40.4
46–65	90	36.0
>65	16	6.4
Total	250	100

multiple comparison. *P* values <0.05 were considered significant while the difference within the group was analyzed using *t* test ($p<0.05$).

Results

Data of 250 malaria patients who are 18 years and above with a complete record for hematological parameters, lipid profile, and renal and hepatic dysfunction parameters from 2016 to 2020 in Sir Yahaya Memorial Hospital Birnin Kebbi, Kebbi State, who met the inclusion criteria were identified. One hundred sixty-three of these were females and 87 were males. That is, females composed 65.2% of malaria patients with complete records while the remaining 34.8% were males (Table 1). Most of the records of the patients contain the malaria test only in most cases. Although the other incomplete records were not used, females still constitute the higher percentage with malaria records in the health facility. For the control, more female records were also available to compare to males (Table 1). Age distribution of the infected patients showed that the infection was more prevalent among the age range 26–45 years which constitute 40.4% of the patients with a complete record followed by the 46–65 years of age bracket. People above 65 years constitute the least infected patients (Table 2).

Hemoglobin concentration, red blood cell counts, and packed cell volume were significantly ($p<0.05$) lower in malaria patients compared to the healthy patients. Moreover, the values were severely affected in the female patients compared to their male counterparts (Table 3). On the other hand, mean corpuscular hemoglobin concentration (MCHC) was significantly higher in patients than in non-malaria patients. It was also higher in female compared to male patients (Table 3). It showed that malaria-induced anemia was more profound in female malaria patients than in male patients. Significant ($p<0.05$) decrease in white blood cell count, platelets count, neutrophils, and monocytes was observed in all malaria patients compared to their respective control (Table 3). Significant ($p<0.05$) increase in lymphocytes was seen in both male and female malaria patients compared to their respective control (Table 3). Significant ($p<0.05$) reduction was more obvious among female malaria patients. Insignificant ($p<0.05$) increase was observed for the eosinophil and the basophil.

Significant increase was observed in serum concentration of AST, ALT, ALP, total bilirubin, and direct bilirubin while a decrease in serum protein, albumin, and glucose

Table 3 Hematological profile of patients with recurrent malaria cases in sir Yahaya memorial hospital Birnin Kebbi from 2016 to 2020

Parameters	Control		Patients	
	Male	Female	Male	female
PCV (%)	42.44 ± 3.81^{a}	39.13 ± 2.73^{b}	37.92 ± 3.82^{c}	29.40 ± 8.96^{d}
RBC ($\times 10^6/\mu L$)	4.22 ± 0.56^{a}	5.9 ± 0.91^{b}	3.34 ± 0.56^{c}	2.9 ± 0.51^{d}
Hemoglobin (g/dl)	13.71 ± 1.22^{a}	13.84 ± 1.31^{a}	10.94 ± 3.11^{b}	8.95 ± 2.80^{c}
MCHC(g/dl)	33.81 ± 1.44^{a}	34.19 ± 1.47^{a}	40.17 ± 3.01^{c}	47.72 ± 2.75^{c}
Total WBC ($\times 10^3/\mu l$)	9.02 ± 3.98^{a}	8.79 ± 3.88^{a}	7.23 ± 1.81^{b}	5.39 ± 1.76^{c}
Neutrophil (%)	58.96 ± 2.47^{a}	58.10 ± 2.97^{a}	45.77 ± 15.27^{b}	42.11 ± 13.34^{b}
Lymphocyte (%)	27.45 ± 2.02^{a}	27.67 ± 1.49^{a}	45.07 ± 18.37^{a}	51.02 ± 15.60^{a}
Monocyte (%)	4.49 ± 1.89^{a}	4.31 ± 2.16^{a}	1.40 ± 1.78^{b}	1.17 ± 1.53^{b}
Eosinophil (%)	1.55 ± 0.67^{b}	1.67 ± 0.77^{b}	1.87 ± 0.73^{b}	1.85 ± 0.75^{b}
Basophil (%)	0.34 ± 0.45^{b}	0.48 ± 0.20^{b}	0.37 ± 0.49^{b}	0.49 ± 0.50^{b}
Platelet ($\times 10^3/\mu L$)	185.0 ± 75.56^{a}	205.0 ± 75.56^{b}	125.93 ± 60.35^{c}	165.93 ± 89.35^{d}

The values are expressed as mean ± standard deviation of the observations in each group

Values with different superscript letters in the same row are significantly different from each other ($P<0.05$)

Table 4 Liver function parameters and serum glucose profiles of patients with recurrent malaria cases in sir Yahaya memorial hospital Birnin Kebbi from 2016 to 2020

Parameters	Control		Patients	
	Male	Female	Male	Female
AST (IU/L)	22.94 ± 10.38^{a}	23.08 ± 11.35^{a}	63.74 ± 19.16^{b}	37.39 ± 17.23^{c}
ALT (IU/L)	28.23 ± 7.98^{a}	28.25 ± 9.56^{a}	52.04 ± 24.19^{b}	35.28 ± 22.04^{c}
ALP (IU/L)	95.11 ± 37.50^{a}	91.84 ± 27.08^{a}	167.40 ± 48.73^{b}	103.77 ± 31.63^{c}
Total protein (g/dL)	7.14 ± 0.72^{a}	7.17 ± 0.76^{a}	5.84 ± 1.43^{b}	5.33 ± 1.70^{b}
Albumin (g/dL)	4.19 ± 0.80^{a}	4.42 ± 0.71^{a}	3.35 ± 2.48^{b}	3.53 ± 2.31^{b}
Total bilirubin (mg/dl)	0.93 ± 0.32^{a}	1.05 ± 0.31^{a}	3.29 ± 1.52^{a}	2.49 ± 1.75^{c}
Direct bilirubin (mg/dl)	0.14 ± 0.09^{a}	0.12 ± 0.08^{a}	0.81 ± 0.48^{b}	0.25 ± 0.17^{c}
Glucose (mmol/l)	5.33 ± 0.57^{a}	5.27 ± 0.65^{a}	3.56 ± 1.85^{b}	2.64 ± 2.03^{c}

The values are expressed as mean ± standard deviation of the observations in each group

Values with different superscript letters in the same row are significantly different from each other ($P<0.05$)

Table 5 renal function of patients with recurrent malaria cases in sir Yahaya memorial hospital Birnin Kebbi from 2016 to 2020

Parameters	Control		Patients	
	Male	Female	Male	Female
Urea (mmol/L)	4.64 ± 1.00^{a}	4.52 ± 0.99^{a}	5.06 ± 2.82^{b}	9.14 ± 3.28^{c}
Creatinine (µmol/L)	72 ± 15^{a}	76 ± 14^{a}	142.5 ± 68.5^{b}	236 ± 154^{c}
Sodium (mmol/L)	138.80 ± 2.30^{a}	138.90 ± 2.79^{a}	130.50 ± 10.65^{a}	106.70 ± 18.15^{b}
Potassium (mmol/L)	3.90 ± 0.79^{a}	4.15 ± 0.69^{a}	3.81 ± 0.60^{a}	2.13 ± 0.78^{c}
Chloride (mmol/L)	100.70 ± 3.80^{a}	100.60 ± 4.04^{a}	97.75 ± 8.49^{a}	75.44 ± 8.81^{a}

The values are expressed as mean ± standard deviation of the observations in each group

Values with different superscript letters in the same row are significantly different from each other ($P<0.05$)

was observed in the malaria patients (Table 4). In malaria infection studies, a simultaneous increase in transaminases and bilirubin is considered as an indicative of liver dysfunction. It seems the effect of the malaria infection on the liver function parameters analyzed correlates positively with the gender. Male tends to show higher dysfunction or compromise in these parameters than female.

The renal function parameters of patients with recurrent malaria showed that there was a significant difference ($p<0.05$) between the serum creatinine concentrations of the malaria-infected patients compared to the control (Table 5). Moreover, the infected female patients also showed a significant elevated serum creatinine concentration compared to the infected males. The same results were observed for the serum urea of the patients compared with the control (Table 5). For the serum electrolytes, there was a significant reduction in sodium, potassium, and chloride level among the female patients compared to the control (Table 5). However, there was no significant ($p<0.05$) difference between the serum electrolytes of male malaria patients and the control (Table 5).

Table 6 Lipid profile of patients with recurrent malaria cases in sir Yahaya memorial hospital Birnin Kebbi from 2016 to 2020

Parameters	Control		Patients	
	Male	Female	Male	Female
Cholesterol (mmol/l)	6.29 ± 0.50^{a}	6.48 ± 0.58^{a}	3.05 ± 1.41^{b}	3.23 ± 2.23^{b}
Triglyceride (mmol/l	1.10 ± 0.77^{a}	1.16 ± 0.86^{a}	2.93 ± 1.15^{a}	3.00 ± 1.14^{a}
HDL (mmol/l)	4.59 ± 1.97^{a}	4.55 ± 2.03^{a}	1.41 ± 0.32^{a}	1.43 ± 0.46^{a}.
LDL (mmol/l)	0.99 ± 0.32^{a}	1.06 ± 0.31^{a}	0.43 ± 0.38^{a}	0.42 ± 0.28^{a}

The values are expressed as mean ± standard deviation of the observations in each group

Values with different superscript letters in the same row are significantly different from each other ($P<0.05$)

The analysis of the patient's data revealed a statistical significant differences in the lipid profile of the patients compared with the control (Table 6). Significant ($P<0.05$) reduction in HDL, LDL, and TC was observed in malaria patients while a significant increase in triacylglycerol

concentrations was found in malaria-infected patients compared to the control (Table 6).

Discussion

From the hospital record, it was observed that prescription of other tests apart from malaria tests is usually recommended for people with recurrent malaria to ascertain the probable cause of treatment failure or other underlining conditions. The number of patients with complete records revealed that females are more likely to seek medical care in health facilities than men. The higher number of women with complete records than men was an indication that more women with recurrent malaria is coming back for follow-up than men. The higher rate of infection among females observed in this study agreed with Sakzabre et al. [21] who observed that females are more susceptible to malaria infection than males. However, Sacomboio et al. [22] and Adedapo et al. [23] observed that the higher prevalence among females may be linked to the fact that pregnant women are more affected by malaria and their inclusion in any study will always result in a higher prevalence among females than males. Since pregnant women are not excluded from the study, the higher rate of infection among women may be due to the inclusion of pregnant women in the study. This is contrary to the findings of Muddaiah and Prakash [24] who observed more cases among males (82%) compared to females (18%) in South Canara, South India, and Yadav et al. [25] who observed higher malaria infection among males (64.1%) than females (34.9%). In the Northern part of Nigeria, males who are the sole provider of the family in majority of the families are likely to be exposed to mosquito bites which increase their tendency of being infected with malaria. However, the tendency to get their medication over the counter than losing man hours in the hospital may be one of the reasons why less records of males were found in the health facility. This tendency has been observed in Saharan Africans by other researchers working with malaria patients [26–29]. In Nigeria, the acceptable adult age is 18 years and the retirement age is 60–65 years depending on the profession. This data shows that malaria mostly affects the active and young population in this region. This may partly explain the level of poverty in this region. It has also been observed from malaria studies in malaria-prone regions that people tend to acquire immunity against malaria over time. Therefore the susceptibility to infection may decrease as the patient grows. This may explain the low infection rate among adults above 60 years [30, 31].

Hematological abnormalities are well known to be the main features of malaria infection [32, 33], especially in *P. falciparum* infection that is common in Africa [34]. The changes observed in malaria infection are known to depend on many factors such as nutritional status, demographic factors, age, genetic susceptibility, hemoglobinopathy in patients, and malarial immunity [11, 35–37]. The study of the malaria patient data showed a significant decrease in hemoglobin concentration, red blood cell counts, and packed cell volume which indicate anemia. This is consistent with previous findings [38, 39]. In addition, an increase in mean corpuscular hemoglobin concentration (MCHC) was observed which is contrary to the other findings but in agreement with the findings of Kotepui et al. [40] who observed that significant reduction of hemoglobin, packed cell volume, and red blood cell counts which indicated anemia among the *P. falciparum*-infected malaria patients did not result in low MCHC as observed by other researchers. They discovered that *P. falciparum* infection resulted in the excessive release of immature red blood cells into blood circulation, which caused an increase in the value of MCHC while the patients were still anemic with a significant reduction of hemoglobin, packed cell volume, and red blood cell counts. In their earlier research, Kotepui et al. [41] also suggested that disease conditions like α^{+}-thalassaemia among malaria patients can also result in the same observation. Anemia observed during malaria infection may be due to the destruction of infected erythrocytes, the removal of parasitized and non-parasitized erythrocytes. Studies on malaria therapy have revealed that for every infected red blood cells destroyed during the treatment of vivax *and falciparum* infection, 32 and 8 non-infected red blood cells respectively are also destroyed [42, 43]. This indiscriminate destruction of erythrocytes resulted from the effect of the antibodies generates against proteins on the red blood cell membrane during malaria infection which does not discriminate between non-parasitized and parasitized red blood cells. This eventually mediates the destruction of non-parasitized and parasitized red blood cells through immune-mediated lysis and phagocytosis [43–47]. The study also observed that the anemia was significantly higher among female malaria patients compared to the male patients making the females more vulnerable to anemia during malaria than the male patients. This was also observed by Sakzabre et al. [21] in their study.

In this study, thrombocytopenia was prevalent among the malaria patients with the female patients having a higher tendency to develop thrombocytopenia compare to the male patients. Similar results have been recorded in malaria research where a statistical significant correlation between malaria infection and thrombocytopenia has been established making thrombocytopenia a reliable predictor and reliable diagnostic marker of malaria infection and severity [31, 38, 41, 48–52]. The

thrombocytopenia observed in malaria infection may be due to excessive removal of platelets by splenic pooling, peripheral destruction of platelets [47], malaria induce shortening of the life span of the platelets [31], antibody (IgG)-mediated platelet destruction [53], destruction by macrophages, platelet aggregation, and oxidative stress [54, 55]. A direct relationship between thrombocytopenia and the level of parasitemia and malaria positivity has also been reported [40, 56]. These make thrombocytopenia and anemia useful diagnostic standards for predicting the severity of malaria infection. This confirmed that females in this area are more prone to malaria than males [51, 57]. There was an increase in lymphocytes, eosinophils, and basophil in the patients but a significant decrease in white blood cells, neutrophils, and monocytes. Leucopenia is commonly associated with malaria infection [39, 48, 53, 58, 59]. Leucopenia is assumed to be due to the splenic sequestration and localization of leucocytes away from the peripheral circulation and other marginal pools rather than actual depletion or stasis [60], but leucocytosis has also been reported [61]. A significant decrease in neutrophils in malaria patients was observed in this study compared to the control. This is contrary to the findings of Maina et al. [39] and Kayode et al. [62].

The increase in lymphocytes observed among the patients in this study contradicts the reduction in lymphocytes observed by Kayode et al. [62] and Wickramasinghe and Abdalla [63].

A decrease in monocytes (monocytopenia) agreed with the findings of Bawah et al. [64] and Srivastava et al. [65] but contrary to the observation of Kotepui et al. [41]. Differences in observation may be due to the immune system of the host, level of parasitemia, severity of the infection, and co-infection.

The data obtained from the hospital records revealed a serious elevation in the liver enzymes and bilirubin in the patients irrespective of their gender compared to the control. This implied malaria-induced liver damage or dysfunction. This agrees with other studies that indicated a direct correlation between malaria and liver dysfunction. Liver dysfunction associated with malaria has been recognized as a major cause of malaria mortality and morbidity [3, 8, 10, 66–69]. Malaria-induced hyperbilirubinemia has been associated with acute kidney injury and pathogenesis of acute renal failure in malaria infection [11–13]. This suggests that hepatic dysfunction is closely related to the development of acute kidney disease. Severe hyperbilirubinemia in malaria infection has been implicated in the pathogenesis of cerebral malaria. Cerebral malaria is significantly associated with mortality in malaria infection, making hyperbilirubinemia a serious concern in malaria patients [12, 16, 17]. Malaria-induced hepatic damage has been linked to malaria-induced intravascular hemolysis induced by free heme overload [70]; systemic inflammation in response to malaria infection that aggravates the host hepatic tissue destruction [71] and the influx of IL-1α-producing neutrophil into the hepatocytes. The main source of IL-1α in the liver during malaria infection has been established to be the infiltrating neutrophils and not the other liver cell populations, though hepatocytes and Kupffer cells produce IL-1α during liver injury [72]. The neutrophils released IL-1α locally to promote the inflammatory response and TNF-α production that initiates apoptosis of hepatocytes [72]. Malaria infection has been shown to downregulate antioxidant gene expression in the liver [73–75]. This results in a reduced ability of the body to clear the free radicals and limit its ability to protect cellular constituents from oxidative damage. Drug-induced damage has been implicated also [76, 77]. Tissue damage leading to the level of serum biomarkers may also be related to reduced oxygen supply and disturbed metabolisms as a result of parasite sequestration in the microvascular capillary system [78]. This made Fazil et al. [67] to recommend liver function tests immediately after malaria is diagnosed in order to ensure early diagnosis of malarial-induced hepatic damage in patients.

This study showed a significant association between malaria infection and hypoglycemia. These findings agreed with the observation of Camara et al. [79] and Willcox et al. [80] who observed a direct link between mortality rate and hypoglycemia. Hypoglycemia in malaria infection is associated with hepatopathy [67]; malaria-induced cytokines [81]; and the use of drugs such as quinine [82], glucose consumption by the parasites during infections [83], and malnutrition [84, 85].

An increase in serum creatinine and urea was observed in the patients. Similar results have been obtained in other malaria studies [86–88]. Elevation of creatinine and urea concentration in the blood of the patients is an indication of possible renal dysfunction and degree of parasitemia in malaria patients [68, 86, 87]. Significant increase in renal disorder biomarkers in malaria patients is associated with vascular lesions triggered by medications, microorganisms, toxins, imbalance in the production of cytokines, endothelial adhesion, and increase glomerular cell proliferation [89–92]. Electrolyte abnormalities serve as an indicator of the severity of malaria in *P. vivax* and *P. falciparum* malaria which are the major species associated with major malaria cases in sub-Saharan African [5, 93]. This abnormality has been linked to malaria-induced acute kidney injury (AKI) [94, 95]. This study showed that malaria infection resulted in a reduction in a serum sodium level (hyponatremia) [5]. Reduction in serum sodium level has been observed in most cases of malaria and has been widely reported in

P. falciparum and *P. vivax* infection, although it is commonly associated with *P. falciparum* malaria than *P. vivax* malaria [93, 96, 97]. These alterations in serum sodium concentration can result in several health conditions [5]. Malaria-induced vasopressin secretion is a major factor that results in the reduction in the concentration of sodium in malaria infection, as sodium is able to gain entry into the infected cells and cause loss of the blood [98]. Potassium imbalance can cause weakness and rapid heartbeat, therefore keeping the potassium level within the normal physiological level is very important [99]. The data of the patients clearly showed a decrease in serum potassium levels due to the malaria infection. This result is similar to the observation of [96]. This has been linked to malaria-induced enhanced urinary excretion of potassium leading to hypokalemia that is known to aggravate the complications associated with malaria infection [100]. A decrease in potassium is more common in *P. falciparum* infection compared to *P. vivax* infection [93].

Electrolyte abnormalities such as hyponatremia have been observed in 30–50% of cases of malaria-induced acute kidney injury (AKI) [94, 95] and are associated with hemolysis and acidosis [94]. Acute kidney injury (AKI) is gradually becoming a public health challenge in Africa due to the increase in the burden of diseases like malaria coupled with the late presentation of malaria patients for treatment and lack of medical resources for efficient care of patients who presented themselves to the health facilities. Acute kidney injury (AKI) has been reported in 1–4.8% of malaria patients in endemic areas and 25–30% in non-immune patients. This contributes to the high mortality rate of around 75% of malaria cases [92, 94, 101]. The autopsy on the kidney of patients who died of *P. falciparum* infection showed that half of the patients died of AKI [92].

Serum total cholesterol, HDL cholesterol, and LDL cholesterol were lower in the patients than in the controls. This is in contrast to some previous studies that reported elevation in cholesterol and these lipoproteins in the blood of malaria patients [102–105]. The reduction in HDL-cholesterol might be due to a decrease in cholesterol transport, inhibition of the liver enzyme by a parasite factor, uptake of the host cholesterol and phospholipids by the parasite, and esterification by lecithin cholesterol acyl transferase [106]. Oxidation of LDL cholesterol has been implicated in its reduction in malaria patients. This has been shown to increase the endothelial expression of adhesion molecules in malaria patients increasing the risk of cardiovascular disease in malaria patients [19]. Reduction in cholesterol levels in malaria patients is in support of earlier reports that hypocholesterolemia was significantly associated with malaria [107–110]. Increases in serum triacylglycerol (TG) were observed in the malaria patients compared to the healthy controls This might be due to increased mobilization of free fatty acids from adipose tissue [111], increases in hepatic fatty acid synthesis, impairment in lipoprotein lipase system, depression of fatty acid oxidation, and derived lipid from the phospholipids of the red blood cell membrane following hemolysis [112, 113].

Conclusion

Liver and kidney function parameters analyzed correlate positively with the gender. Infected males tend to show higher dysfunction in liver parameters while infected female patients showed a significant elevation in kidney dysfunction parameters. The positive correlation between gender and liver and kidney dysfunction needs to be analyzed further to see if other factors are involved. The result of this study showed that there is a danger of potential widespread acute renal and hepatic failure with the attendant morbidity and mortality. It is therefore recommended that liver and kidney function tests be recommended for patients with recurrent malaria and patients with failed self-medications in the endemic region to ensure early diagnosis of malarial-induced kidney and liver injury in malaria patients.

Abbreviations

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; ALP: Alkaline phosphatase; PCV: Packed cell volume; MCHC: Mean corpuscular hemoglobin concentration; RBC: Red blood cell; WBC: White blood cell; TC: Total cholesterol.

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Consent to participate

Not applicable, since the study is retrospective.

Authors' contributions

ORJ designed the study; ORJ and JIG collected the data; ORJ, JIG, ADM, GG, and LIY analyzed and interpreted the data; ORJ and JIG wrote the first draft manuscript; ORJ, GG, ADM, and LIY performed the critical revisions of the manuscript; LIY, ADM, and GG provided the administrative, technical, and material support; ORJ coordinated and supervised the study. The the authors read and approved the final manuscript.

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Availability of data and materials

The data used to support the findings of this study are available on request from the corresponding author.

Declarations

Ethics approval and consent to participate

Ethical approval was obtained from the hospital management before the commencement of data collection. The risk of loss of confidentiality was eliminated by the removal of personal identifiers and the use of identification numbers. All ethical procedures involving the use of human subjects under clinical settings were duly followed. The retrospective data was collected after ethical approval (SYMHBK/SUB/17/VOL.III/196) was obtained.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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